percent, you are going to pay 10 percent in interest. That meant that her monthly payments went up $200 a month. A Member of Congress may not miss $200 a month, but Ms. McGee will. The monthly payment which she is now required to make will take all of the money that is sent to her in her Social Security check. She is about to lose that home. After 10 years of living her dream, she is about to lose it. She is one of the victims we are talking about, because of the resetting of an adjustable rate mortgage.

One would hope Ms. McGee is the kind of person to be helped by the administration's suggestion on mortgages, but sadly, she is not. She wouldn't qualify, and that is sad. It tells you that this is a safety net that has too big a hole in it and that a lot of poor people are going to fall through.

I have a plan that will go further than the Bush administration plan. I want to change the bankruptcy laws for about a fourth of the people facing foreclosure who end up in bankruptcy court. I want to give them a chance. If they have enough income, the court can order changing the terms of the mortgage, the interest rate and the principal, no lower than the fair market value of the property as of the time of the bankruptcy, and by renegotiating the terms, the people may be able to stay in their homes.

What happens if the proposal I have made doesn't become law? Well, there will be a real foreclosure. They will have to leave their homes. Their homes will be sold on the market. For the lender, what does it mean when you go through foreclosure? It means $50,000 in debts from the foreclosure process. It also means facing the possibility—the very real possibility—that you are going to lose 20 to 30 percent of the value of the loan in a foreclosure sale.

That is the reality, and I hope we can change it. I hope that what we call a mortgage crisis today will become a crisis we respond to as a nation on a bipartisan basis: Congress and the President helping the American people realize their American dreams, live in their homes, and not see the value of their neighborhoods diminish.

Mr. President, I see Senator BROWNBACK is here. I yield the floor.

The PRESIDING OFFICER. The Senator from Kansas is recognized.

Mr. BROWNBACK. Mr. President, I ask unanimous consent to speak as in morning business.

The PRESIDING OFFICER. We are in morning business. Without objection, it is so ordered.

STEM CELL RESEARCH

Mr. BROWNBACK. Mr. President, I rise to discuss a recent enormous scientific breakthrough on a topic that has engaged this body for much of the past 8 years. I think this is a day that many of us—I think perhaps all of us—have hoped would take place. I ask unanimous consent to include in the RECORD at the end of my remarks an article that broke lose right around Thanksgiving.

The PRESIDING OFFICER. Without objection, it is so ordered.

(See exhibit 1.)

Mr. BROWNBACK. Mr. President, this article is by Dr. James Thomson, University of Wisconsin. Some may recognize that name. His name has been used on this floor many times during the past 8 years on the issue of embryonic stem cell research. He is the man who discovered human embryonic stem cells about 10 years ago and described them as being what is called pluripotent, which means that an embryonic stem cell could form any other type of cell tissue in the body, whether it is for the eye, brain, bone, or skin. Any type of cell tissue could regenerate on a fast basis, and it was thought that these sorts of pluripotent embryonic stem cells were going to solve a number of our human health problems. Many of my colleagues on both sides of the aisle embraced the news and said this is a fabulous thing and we are going to be able to now cure a number of people from diseases who have had great problems and difficulties, and we want cures for them.

There was an ethical glitch with it in that it took the destruction of a human embryo to get these human embryonic stem cells, and therein ensued a fight that engaged the country and engaged the world about the tension between cures and an ethical recognition of human life and the sacredness of human life. It has been a long debate. I am hopeful that the article I submitted into the RECORD is the bookend on the other end of this debate that was started by Professor Thomson and that, in many respects, I hope is ended by Professor Thompson and his colleagues.

In this article they describe a new type of pluripotent stem cell that is manipulated by man. They call it an induced pluripotent stem cell. This is an elegant and simple process where they take a skin cell from an individual and they reprogram it to be able to act like an embryonic stem cell, or what they call an induced pluripotent stem cell. They then are able to get it to generate more embryonic-like stem cells that are pluripotent and which then can be used to treat diseases or to study diseases, thus removing the need to develop and have a human embryo destroyed, or the origination of the embryonic stem cells, thus removing the problem of not being able to get a genetic match so that we have to go to a cloned embryonic stem cell, or a cloned human to create an embryonic stem cell that matches genetically. You don't have to do that. Get a person's skin cells, reprogram them, back in, pluripotent, to form any type of cell—elegant, simple.

There are still many barriers to go on embryonic-like stem cells anyway because they have had a problem with tumor formation. But on the ethical issue, I am hopeful we are on the other bookend, and it is now over; that we don't need to destroy young human life for cures; that we don't need to destroy them for pluripotent cells; that we can do it much simpler and ethically and that good ethics is good science.

I put a description up here of what Dr. Thomson said on this subject. There was a University of Tokyo professor who came out with an article the same day, using a slightly different or modified technique, to be able to do this in humans. The University of Tokyo professor had done this earlier in mice and now has perfected it in human cells. He came out saying the same thing:

> These induced pluripotent cells described here meet the defining criteria we originally proposed for human ES cells, with the significant exception that the induced pluripotent cells are not derived from embryos.

That was Dr. James Thomson.

I want to speak about this to my colleagues because we have had so many debates on the Senate floor about this topic. I hope my colleagues will research this. A number of people in the scientific field are saying: Great, but let's not stop embryonic stem cell work and destroying embryos for research purposes. Or let's not stop human cloning because it appears now that the only reason to clone a human would be to bring a human to live birth at this point in time, which still has everybody in this body opposed to that type of human cloning.

It is noteworthy that the ''father'' of Dolly the sheep has said he has given up on human cloning to go to this type of technique rather than human cloning to provide these sorts of cures and research.

Mr. President, I also ask unanimous consent to be printed in the RECORD at the end of my comments a Telegraph article from the United Kingdom in which Ian Wilmut announced he is shunning human cloning.

The PRESIDING OFFICER. Without objection, it is so ordered.

(See exhibit 2.)

Mr. BROWNBACK. Mr. President, it is my hope that we can move together in finding cures and developing research that cures humans that is ethical and sound and doesn't destroy young human life.

We have been able to do quite a bit of this already. We recently found there was scientific work done by a Northwestern University professor in developing cures and treatments for type I diabetes using stem cells. Again, this is adult stem cells, which is ethical and moral, no problem with it. The only problem I found with it is that the Northwestern professor was having to do this in Brazil rather than in the United States to get support and funding. He is saying this:

> Though too early to call it a cure, the procedure has enabled the young people, who have type I diabetes, to live insulin-free so

far, some as long as 3 years. The treatment involves stem cell transplants from the patient's own blood.

For parents who are dealing with juvenile diabetes and those difficulties, this is fabulous news in humans. We need more of it, and we need it to take place in the United States and not Brazil. Nothing against Brazil. I am glad for it to take place there, but I want it here for our children. We now have—as I have said previously on the floor—73 different human applications for adult stem cells. We have not been able to come up with any in the embryonic field yet. I think a bigger number—and we will verify this for my colleagues, as it is not verified yet—is somewhere north of 400,000 people who are now being treated with adult or cord blood stem cells in the United States and different places around the world, the majority being U.S. citizens. Of course, we don't have any in the embryonic field because it continues to struggle with tumor formation as an issue. These are wonderful numbers of treatments that we are getting in different human maladies and, hopefully, we can verify that number of 400,000 people being treated with stem cells, getting heart tissue and spinal cord tissue to regenerate, and Parkinson's treatment is coming forward. This is a beautiful set of treatments—all ethical.

I want to look at the budgetary numbers briefly to remind my colleagues where we have invested taxpayer funding in this field. It is my hope that as we look at the numbers—we have an ethical issue on human embryonic stem cell research, and I believe we have crossed over the line. I hope we can continue to look at our funding issues, where we are putting a lot of money, and have put a lot of money, into embryonic stem cell research. We are looking at $140 million in fiscal year 2006 and over half a billion since 2002 in embryonic stem cell research of both human and nonhuman types. We have not cured a single patient yet with that money.

May I submit to my colleagues that with over half a billion dollars, we could be treating and developing these cures in the United States and not in Brazil.

In trying to set aside all of the sharp edges that have now been associated with this debate, and focusing just on patients and treating people, I hope we will say we are all in this for cures, for treating people. So if I could take portions of these funds and put it into treating people and getting more people treated for Parkinson's, congestive heart failure, or diabetes—all the things that we are actually doing in humans today but that need more research in funding—that we would say: OK, you are right. We don't have to go the embryonic stem cell route now. Let's go to where people are getting treated and treat people.

This is about curing people. That is what we have debated and talked about for some period of time, curing people. We have one that is working and one that doesn't. Yet we have invested pretty heavily in this.

I ask my colleagues if there is some way that we could put the swords down and talk about this rationally, stop the fighting and say how do we treat people. I believe that is our objective.

With that, I thank my colleagues for their indulgence in this debate. It will continue to come up. The next issue will be human animal crosses. I advise my colleagues on this, you will see people pushing to cross genetic materials from animals into humans. They are going to say it is going to cure a lot of people. I think it is an enormous ethical boundary that we should not cross at this point in time, with our understanding of life and what it is to be human. I hope before we go that route, we will all get together and say we are going to pause for a while on this one. This is too big for all of us, and we want to think about this for a while—left, right, middle. We have a ways to go to get some cures. We are getting them. We don't need to cross over to that. We can think about that.

I yield the floor.

EXHIBIT 1

INDUCED PLURIPOTENT STEM CELL LINES DERIVED FROM HUMAN SOMATIC CELLS

Somatic cell nuclear transfer allows transacting factors present in the mammalian oocyte to reprogram somatic cell nuclei to an undifferentiated state. Here we show that four factors (OCT4, SOX2, NANOG, and LIN28) are sufficient to reprogram human somatic cells to pluripotent stem cells that exhibit the essential characteristics of embryonic stem cells. These human induced pluripotent stem cells have normal karyotypes, express telomerase activity, express cell surface markers and genes that characterize human ES cells, and maintain the developmental potential to differentiate into advanced derivatives of all three primary germ layers. Such human induced pluripotent cell lines should be useful in the production of new disease models and in drug development as well as application in transplantation medicine once technical limitations (for example, mutation through viral integration) are eliminated.

Mammalian embryogenesis elaborates distinct developmental stages in a strict temporal order. Nonetheless, because development is dictated by epigenetic rather than genetic events, differentiation is, in principle, reversible. The cloning of Dolly demonstrated that nuclei from mammalian differentiated cells can be reprogrammed to an undifferentiated state by trans-acting factors present in the oocyte (1), and this discovery led to a search for factors that could mediate similar reprogramming without somatic cell nuclear transfer. Recently. four transcription factors (Oct4, Sox2, c-myc, and Klf4) were shown to be sufficient to reprogram mouse fibroblasts to undifferentiated, pluripotent stem cells (termed induced pluripotent stem (iPS) cells) (2–5). Reprogramming human cells by defined factors would allow the generation of patient-specific pluripotent cell lines without somatic cell nuclear transfer, but the observation that the expression of c-Myc causes death and differentiation of human ES cells suggests that combinations of factors lacking this gene are required to reprogram human cells (6). Here we demonstrate that OCT4, S0X2, NANOG, and LIN28 are sufficient to reprogram human somatic cells.

Human ES cells can reprogram myeloid precursors through cell fusion (7). To identify candidate reprogramming factors, we compiled a list of genes with enriched expression in human ES cells relative to myeloid precursors, and prioritized the list based on known involvement in the establishment or maintenance of pluripotency (table S1). We then cloned these genes into a lentiviral vector (fig. S1) to screen for combinations of genes that could reprogram the differentiated derivatives of an OCT4 knock-in human ES cell line generated through homologous recombination (8). In this cell line, the expression of neomycin phosphotransferase, which make cells resistant to geneticin, is driven by an endogenous OCT4 promoter, a gene that is highly expressed in pluripotent cells but not in differentiated cells. Thus reprogramming events reactivating the OCT4 promoter can be recovered by geneticin selection. The first combination of 14 genes we selected (table S2) directed reprogramming of adherent cells derived from human ES cell-derived CD45+ hematopoietic cells (7, 9), to geneticin-resistant (OCT4 positive) colonies with an ES cell-morphology (fig. S2A) (10). These geneticin-resistant colonies expressed typical human ES cell-specific cell surface markers (fig. S2B) and formed teratomas when injected into immunocompromised SCID-beige mice (fig. S2C).

By testing subsets of the 14 initial genes. we identified a core set of 4 genes, OCT4, SOX2, NANOG, and LIN28, that were capable of reprogramming human ES cell-derived somatic cells with a mesenchymal phenotype (Fig. 1A and fig. S3). Removal of either OCT4 or SOX2 from the reprogramming mixture eliminated the appearance of geneticin resistant (OCT4 positive) reprogrammed mesenchymal clones (Fig. 1A). NANOG showed a beneficial effect in clone recovery from human ES cell-derived mesenchymal cells but was not required for the initial appearance of such clones (Fig. 1A). These results are consistent with cell fusion-mediated reprogramming experiments, where overexpression of Nanog in mouse ES cells resulted in over a 200-fold increase in reprogramming efficiency (11). The expression of NANOG also improves the cloning efficiency of human ES cells (12). and thus could increase the survival rate of early reprogrammed cells. LIN28 had a consistent but more modest effect on reprogrammed mesenchymal cell clone recovery (Fig. 1A).

We next tested whether OCT4, SOX2, NANOG, and LIN28 are sufficient to reprogram primary, genetically unmodified, diploid human fibroblasts. We initially chose IMR90 fetal fibroblasts because these diploid human cells are being extensively characterized by the ENCODE Consortium (13), are readily available through the American Type Culture Collection (ATCC, Catalog No. CCL–186) and have published DNA fingerprints that allow confirmation of the origin of reprogrammed clones. IMR90 cells also proliferate robustly for more than 20 passages before undergoing senescence but grow slowly in human ES cell culture conditions, a difference that provides a proliferative advantage to reprogrammed clones and aids in their selection by morphological criteria (compact colonies, high nucleus to cytoplasm ratios, and prominent nucleoli) alone (14, 15). IMR90 cells were transduced with a combination of OCT4, SOX2, NANOG, and LIN28. Colonies with a human ES cell morphology (iPS colonies) first became visible after 12 days posttransduction. On day 20, a total of 198 iPS colonies were visible from 0.9 million starting IMR90 cells whereas no iPS colonies were observed in non-

transduced controls. Forty-one iPS colonies were picked, 35 of which were successfully expanded for an additional three weeks. Four clones (iPS(IMR90)1–4) with minimal differentiation were selected for continued expansion and detailed analysis.

Each of the four iPS(IMR90) clones had a typical human ES cell morphology (Fig. 1B) and a normal karyotype at both 6 and 17 weeks of culture (Fig. 2A). Each iPS(IMR90) clone expressed telomerase activity (Fig. 2B) and the human ES cell-specific cell surface antigens SSEA–3, SSEA–4, Tra–1–60 and Tra–1–81 (Fig. 2C) whereas the parental IMR90 cells did not. Microarray analyses of gene expression of the four iPS(IMR90) clones confirmed a similarity to five human ES cell lines (H1, H7, H9, H13 and H14) and a dissimilarity to IMR90 cells (Fig. 3, table S3, and fig. S4). Although there was some variation in gene expression between different iPS(IMR90) clones (fig. S5), the variation was actually less than that between different human ES cell lines (Fig. 3A and table S3). For each of the iPS(IMR90) clones, the expression of the endogenous OCT4 and NANOG was at levels similar to that of human ES cells, but the exogenous expression of these genes varied between clones and between genes (Fig. 3B). For OCT4, some expression from the transgene was detectable in all of the clones, but for NANOG, most of the clones demonstrated minimal exogenous expression, suggesting silencing of the transgene during reprogramming. Analyses of the methylation status of the OCT4 promoter showed differential methylation between human ES cells and IMR90 cells (fig. S6). All four iPS(IMR90) clones exhibited a demethylation pattern similar to that of human ES cells and distinct from the parental IMR90 cells. Both embryoid body (fig. S7) and teratoma formation (Fig. 4) demonstrated that all four of the reprogrammed iPS(IMR90) clones had the developmental potential to give rise to differentiated derivatives of all three primary germ layers. DNA fingerprinting analyses (short tandem repeat-STR) confirmed that these iPS clones were derived from IMR90 cells and confirmed that they were not from the human ES cell lines we have in the laboratory (table S4). The STR analysis published on the ATCC website for IMR90 cells employed the same primer sets and confirms the identity of the IMR90 cells used for these experiments. The iPS(IMR90) clones were passaged at the same ratio (1:6) and frequency (every 5 days) as human ES cells, had doubling times similar to that of the human H1 ES cell line assessed under the same conditions (table S5), and as of this writing, have been in continuous culture for 22 weeks with no observed period of replicative crisis. Starting with an initial 4 wells of a 6-well plate of iPS cells (one clone/well, approximately 1 million cells), after 4 weeks of additional culture, 40 total 10-cm dishes (representing approximately 350 million cells) of the 4 iPS(IMR90) clones were cryopreserved and confirmed to have normal karyotypes.

Since IMR90 cells are of fetal origin, we next examined reprogramming of postnatal fibroblasts. Human newborn foreskin fibroblasts (ATCC, Catalog No. CRL–2097) were transduced with OCT4, SOX2, NANOG, and LIN28. From 0.6 million foreskin fibroblasts, we obtained 57 iPS colonies. No iPS colonies were observed in non-transduced controls. Twenty-seven out of 29 picked colonies were successfully expanded for three passages, four of which (iPS(foreskin)-1 to 4) were selected for continued expansion and analyses. DNA fingerprinting of the iPS(foreskin) clones matched the fingerprints for the parental fibroblast cell line published on the ATCC website (table S4).

Each of the four iPS(foreskin) clones had a human ES cell morphology (fig. S8A), had a normal karyotype (fig. S8B), and expressed telomerase, cell surface markers, and genes characteristic of human ES cells (Figs. 2 and 3 and fig. S5). Each of the four iPS(foreskin) clones proliferated robustly, and as of this writing, have been in continuous culture for 14 weeks. Each clone demonstrated multilineage differentiation both in embryoid bodies and teratomas (figs. S9 and S10); however, unlike the iPS(IMR90) clones, there was variation between the clones in the lineages apparent in teratomas examined at 5 weeks. In particular, neural differentiation was common in teratomas from iPS(foreskin) clones 1 and 2 (fig. S9A), but was largely absent in teratomas from iPS(foreskin) clones 3 and 4. Instead, there were multiple foci of columnar epithelial cells reminiscent of primitive ectoderm (fig. S9D). This is consistent with the embryoid body data (fig. Sl0), where the increase in PAX6 (a neural marker) in iPS(foreskin) clones 3 and 4 was minimal compared to the other clones, a difference that correlated with a failure to downregulate NANOG and OCT4. A possible explanation for these differences is that specific integration sites in these clones allowed continued high expression of the lentiviral transgenes, partially blocking differentiation.

PCR for the four transgenes revealed that OCT4, SOX2, and NANOG were integrated into all four of the iPS(IMR90) clones and all four of the iPS(foreskin) clones, but that LIN28 was absent from one iPS(IMR90) clone (#4) and from one iPS(foreskin) clone (#1) (Fig. 2D). Thus, although LIN28 can influence the frequency of reprogramming (Fig. 1A), these results confirm that it is not absolutely required for the initial reprogramming, nor is it subsequently required for the stable expansion of reprogrammed cells.

The human iPS cells described here meet the defining criteria we originally proposed for human ES cells (14), with the significant exception that the iPS cells are not derived from embryos. Similar to human ES cells, human iPS cells should prove useful for studying the development and function of human tissues, for discovering and testing new drugs, and for transplantation medicine. For transplantation therapies based on these cells, with the exception of autoimmune diseases, patient-specific iPS cell lines should largely eliminate the concern of immune rejection. It is important to understand, however, that before the cells can be used in the clinic, additional work is required to avoid vectors that integrate into the genome, potentially introducing mutations at the insertion site. For drug development, human iPS cells should make it easier to generate panels of cell lines that more closely reflect the genetic diversity of a population, and should make it possible to generate cell lines from individuals predisposed to specific diseases. Human ES cells remain controversial because their derivation involves the destruction of human preimplantation embryos and iPS cells remove this concern. However, further work is needed to determine if human iPS cells differ in clinically significant ways from ES cells.

EXHIBIT 2

DOLLY CREATOR PROF IAN WILMUT SHUNS CLONING

(By Roger Highfield)

The scientist who created Dolly the sheep, a breakthrough that provoked headlines around the world a decade ago, is to abandon the cloning technique he pioneered to create her.

Prof Ian Wilmut's decision to turn his back on ''therapeutic cloning'', just days after US researchers announced a breakthrough in the cloning of primates, will send shockwaves through the scientific establishment.

He and his team made headlines around the world in 1997 when they unveiled Dolly, born July of the year before.

But now he has decided not to pursue a licence to clone human embryos, which he was awarded just two years ago, as part of a drive to find new treatments for the devastating degenerative condition, Motor Neuron disease.

Prof Wilmut, who works at Edinburgh University, believes a rival method pioneered in Japan has better potential for making human embryonic cells which can be used to grow a patient's own cells and tissues for a vast range of treatments, from treating strokes to heart attacks and Parkinson's, and will be less controversial than the Dolly method, known as ''nuclear transfer.''

His announcement could mark the beginning of the end for therapeutic cloning, on which tens of millions of pounds have been spent worldwide over the past decade. ''I decided a few weeks ago not to pursue nuclear transfer,'' Prof Wilmut said.

Most of his motivation is practical but he admits the Japanese approach is also ''easier to accept socially.''

His inspiration comes from the research by Prof Shinya Yamanaka at Kyoto University, which suggests a way to create human embryo stem cells without the need for human eggs, which are in extremely short supply, and without the need to create and destroy human cloned embryos, which is bitterly opposed by the pro life movement.

Prof Yamanaka has shown in mice how to turn skin cells into what look like versatile stem cells potentially capable of overcoming the effects of disease.

This pioneering work to revert adult cells to an embryonic state has been reproduced by a team in America and Prof Yamanaka is, according to one British stem cell scientist, thought to have achieved the same feat in human cells.

This work has profound significance because it suggests that after a heart attack, for example, skin cells from a patient might one day be manipulated by adding a cocktail of small molecules to form muscle cells to repair damage to the heart, or brain cells to repair the effects of Parkinson's. Because they are the patient's own cells, they would not be rejected.

In theory, these reprogrammed cells could be converted into any of the 200 other type in the body, even the collections of different cell types that make up tissues and, in the very long term, organs too. Prof Wilmut said it was ''extremely exciting and astonishing'' and that he now plans to do research in this area.

This approach, he says, represents, the future for stem cell research, rather than the nuclear transfer method that his large team used more than a decade ago at the Roslin Institute, near Edinburgh, to create Dolly.

In this method, the DNA contents of an adult cell are put into an emptied egg and stimulated with a shock of electricity to develop into a cloned embryo, which must be then dismantled to yield the flexible stem cells.

More than a decade ago, biologists though the mechanisms that picked the relevant DNA code that made a cell adopt the identity of skin, rather than muscle, brain or whatever, were so complex and so rigidly fixed that it would not be possible to undo them.

They were amazed when this deeply-held conviction was overturned by Dolly, the first mammal to be cloned from an adult cell, a feat with numerous practical applications, most remarkably in stem cell science.

But although ''therapeutic cloning'' offers a way to get a patient's own embryonic stem cells to generate unlimited supplies of cells

and tissue there is an intense search for alternatives because of pressure from the pro-life lobby, the opposition of President George W Bush and ever present concerns about cloning babies.

Prof Wilmut's decision signals the lack of progress in extending his team's pioneering work on Dolly to humans.

The hurdles seem to have been overcome a few years ago by a team led by Prof Hwang Woo-Suk in South Korea, with whom he set up a collaboration.

Then it was discovered Prof Hwang's work was fraudulent. ''We spent a long time talking to him before discovering it was all a fraud,'' he said. ''I never really got started again after that.''

And Prof Wilmut believes there is still a long way to go for therapeutic cloning to work, despite the headlines greeting this week's announcement in Nature by Dr Shoukhrat Mitalipov and colleagues at Oregon Health & Science University, Beaverton, that they cloned primate embryos.

In all Dr Mitalipov used 304 eggs from 14 rhesus monkeys to make two lines of embryonic stem cells, one of which was chromosomally abnormal. Dr Mitalipov himself admits the efficiency is low and, though his work is a ''proof of principle'' and the efficiency of his methods has improved, he admits it is not yet a cost effective medical option.

Cloning is still too wasteful of precious human eggs, which are in great demand for fertility treatments, to consider for creating embryonic stem cells. ''It is a nice success but a bit limited,'' commented Prof Wilmut. ''Given the low efficiency, you wonder just how long nuclear transfer will have a useful life.''

Nor is it clear, he said, why the Oregon team was successful, which will hamper attempts to improve their methods. Instead, Prof Wilmut is backing direct reprogramming or ''de-differentiation'', the embryo free route pursued by Prof Yamanaka, which he finds ''100 times more interesting.''

''The odds are that by the time we make nuclear transfer work in humans, direct reprogramming will work too.

I am anticipating that before too long we will be able to use the Yamanaka approach to achieve the same, without making human embryos. I have no doubt that in the long term, direct reprogramming will be more productive, though we can't be sure exactly when, next year or five years into the future.''

Prof Yamanaka's work suggests the dream of converting adult cells into those that can grow into many different types can be realised remarkably easily.

When his team used a virus to add four genes (called Oct4, Sox2, c-Myc and KIf4) into adult mouse fibroblast cells they found they could find resulting embryo-like cells by sifting the result for the one in 10,000 cells that make proteins Nanog or Oct4, both typical markers of embryonic cells.

When they studied how genes are used in these reprogrammed cells, ''called induced pluripotent stem (iPS) cells'', they were typical of the activity seen in an embryo. In the test tube, the new cells look and grow like embryonic stem cells.

And they were also able to generate viable chimaeras from the cells, where the embryo cells created by the new method could be mixed with those of a mouse embryo to grow into a viable adult which could pass on the DNA of the reprogrammed cells to the next generation.

Nonetheless, there will have to be much work to establish that they behave like embryo cells, let alone see if they are safe enough to use in the body. Even so, in the short term they will offer an invaluable way to create lines of cells from people with serious diseases, such as motor neuron disease, to shed light on the mechanisms.

Given the history of fraud in this field, the Oregon research was reproduced by Dr David Cram and colleagues at Monash University, Melbourne. ''At this stage, nuclear transfer to create pluripotent stem cell lines remains an inefficient process,'' said Dr Cram.

Mr. BROWNBACK. Mr. President, I suggest the absence of a quorum.

The PRESIDING OFFICER. The clerk will call the roll.

The legislative clerk proceeded to call the roll.

Mr. CRAIG. Mr. President, I ask unanimous consent that the order for the quorum call be rescinded.

The PRESIDING OFFICER (Mr. SALAZAR). Without objection, it is so ordered.

Mr. CRAIG. Mr. President, let me inquire, we are in morning business?

The PRESIDING OFFICER. The Senator is correct.

ALTERNATIVE MINIMUM TAX AND 3-PERCENT WITHHOLDING

Mr. CRAIG. Mr. President, I come to the floor of the Senate today to speak about two very important issues to America's taxpayers.

The first, of course, is the alternative minimum tax on which we had a cloture vote this morning. That is a very serious matter. I voted against a motion to proceed because I do not believe the best way to prevent a tax increase on 25 million taxpayers is to raise taxes elsewhere by about $80 billion. There is an old phrase out there saying that you are going to rob Peter to pay Paul. Obviously, Peter feels his pocket has been picked, but Paul might feel pretty good about it. And that is the scheme that was played out here. It is a switch game that goes on. The alternative minimum tax is important, but you don't do what they are doing. How can you give a tax break that is already going out somewhere else and raising taxes to give it? That is the issue at hand. I hope the majority is serious about protecting millions of middle-class taxpayers by bringing realistic, bipartisan legislation to fix the AMT, something both sides of the aisle can and, in all fairness, should support.

Even though I did not support how this legislation was crafted, there is a provision in the tax extender package that I wish to highlight because it is very important to taxpayers.

The bill we just voted on contained a provision to delay for 1 year a Federal mandate that requires every level of government—Federal, State, and local—to deduct and withhold a 3-percent tax on all payments of goods and services if that government spends $100 million or more for those goods and services. Oh, yes, that is a shuffle game that has been going on in the Finance Committees in the House and the Senate for some time, and it was slipped in as a way to grab some money. I saw that coming early on and began to object to it and began to look at the figures on it when others of us were saying: Well, gee, I thought that was an ability to raise some more money. I was pleased this issue was finally addressed, but what we need is full repeal of this terrible tax policy, not just a 1-year delay, although I must say a 1-year delay is going to awaken a lot of my colleagues because their State, county, and city governments are going to be calling, if they haven't already, saying: Wake up, you are putting a substantial tax on top of us.

I have come to the floor of the Senate today to renew a promise I made over a year ago. The same day this Senate provided tax relief for millions of Americans by passing the Tax Increase Prevention and Reconciliation Act of 2005, for which I voted, I pledged to do all I could to remove this terrible provision I just talked about that was quietly slipped into the conference report as a last-minute revenue raiser. So I stand here today to renew that pledge.

Last year, I told Members of the Senate this provision would not go unnoticed, and I was right. Once taxpayers learned what this Congress had done in the middle of the night when somebody wasn't watching, they began to react. Angry taxpayers from across the Nation are joining forces, organizing coalitions, and rallying grassroots support to fix this unjust tax policy. I applaud them for their efforts, and I am here to help them.

Let me take a couple of minutes to share what hundreds of angry taxpayers shared with me. I want every Member of the Senate to listen carefully. I want them to understand how this 3-percent tax withholding will affect each and every one of their constituents. I want them to understand why this mandatory 3-percent withholding tax is so bad.

First, 3-percent withholding was justified in the name of closing a tax gap. Proponents argued it would improve compliance. I will show a chart. They say it will improve tax compliance by approximately $7 billion over 5 years. I do not agree, and neither do the numbers.

These numbers are based on the Joint Tax Committee's original estimates. These numbers are simply slightly different when we take the 1-year delay that was in the provision that was on the floor this morning into account. But these numbers tell the story of why this is such a terrible provision.

In 2011, the first year this provision goes into effect, this 3-percent withholding tax accounts for about $6.79 billion in new revenue—boom, a big chunk of new revenue. Can't you see the spenders on the floor of the Senate salivating as they factor that into their budgets and bring down their deficit margins? However, each year after this provision only brings in about $200 million. Why is that? I will tell you. Because about $5.8 billion will be rightly returned to the taxpayers each year